

A case report on the delayed diagnosis of transverse myelitis in a 61-year-old male farmer

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Transverse myelitis is a neurological disorder that results in acute focal inflammation of the spinal cord. It can present with a varied spectrum of neurological signs and symptoms which can make diagnosing a challenge, and delayed diagnosis a frequent complication. This is a case of a 61-year-old male who presented with back pain complicated by neurological symptoms that should have warranted immediate referral to a neurologist. It took approximately five weeks from the onset of his symptoms to be referred to a neurologist, and a further four months to the diagnosis of transverse myelitis. The authors hope to stress the importance of thorough evaluations including neurological exams when new symptoms present and to emphasize regular interprofessional collaboration, that may have prevented the delay in diagnosis seen in this case.

(JCCA. 2020;64(2):131-138)

KEY WORDS: collaboration, diagnosis, management, myelitis, myelopathy, neuropathy

La myélite transverse est un trouble neurologique se manifestant par une inflammation focale aiguë de la moelle épinière. Le sujet peut présenter divers signes et symptômes neurologiques qui peuvent rendre le diagnostic difficile. Un diagnostic tardif entraîne de fréquentes complications. Il s'agit d'un homme de 61 ans ayant des dorsalgies et des symptômes neurologiques qui auraient dû justifier son renvoi immédiat à un neurologue. Environ cinq semaines après l'apparition des symptômes se sont écoulées avant le renvoi à un neurologue, et par la suite quatre mois se sont écoulés avant qu'un diagnostic de myélite transverse ne soit établi. Les auteurs de l'étude espèrent souligner l'importance des évaluations poussées, y compris des examens neurologiques lorsque de nouveaux symptômes apparaissent, et l'importance d'une collaboration régulière entre professions, ce qui aurait permis d'éviter le retard de diagnostic dans le cas de ce patient.

(JCCA. 2020;64(2):131-138)

MOTS CLÉS : collaboration, diagnostic, gestion, myélite, myélopathie, neuropathie

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The authors have no disclaimers, competing interests, or sources of support or funding to report in the preparation of this manuscript. The involved patient's wife provided consent for case publication.

Introduction

Transverse myelitis (TM) is defined as a neurological disorder of focal inflammation in the spinal cord that can result in motor, sensory, and autonomic dysfunction below the level of the lesion.^{1,2} It is considered rare, with a prevalence of one to eight people per million, and a yearly incidence of 1400 in the United States.¹ As with many spinal cord pathologies, morbidity is common.¹ While TM has no race, genetic, or geographic predispositions, it is bimodal in classic age presentation, appearing in the second or fourth decades.¹ Like multiple sclerosis (MS), it tends to affect females more than males.¹ It is considered more common if it is acquired after the diagnosis of MS or neuromyelitis optica.⁴ TM can be diagnosed as primary or idiopathic in nature, or secondary to another disease. There is debate, however, as to whether TM is truly a primary disease or if it is always secondary to another disease process. Some suggest that >50% of all cases are the result of an infection (such as a flare up of varicella zoster, cytomegalovirus, Epstein-Barr or influenza) that preceded the presentation of symptoms.^{1,3} Further, it is often difficult to establish the preceding infection or cause; as such, up to 60% of TM cases are diagnosed as idiopathic, with the understanding the antecedent cause may be missed.³ There are three main categories of differential diagnoses for TM, including demyelination (ie MS, neuromyelitis

optica, and idiopathic transverse myelitis), infection (ie varicella zoster, herpes simplex virus), and inflammatory autoimmune disorders (ie systemic lupus erythematosus, neurosarcoidosis).⁵ There is some suggestion that this disease is not a purely demyelinating disorder, but a mix of all three.⁶ Given this, diagnostic accuracy is challenging.⁶ There are a few suggested mechanisms of neural injury in the process of TM⁷: the bystander effect, molecular mimicry, and humoral response. These mechanisms can result in compressive or non-compressive spinal cord injury.

The bystander effect results in damage to the spinal cord through direct or indirect interaction of the microbial infection and the immune-mediated response against the agent. Molecular mimicry is the process of the body creating antibodies to a bacterial cross-reactive antigen causing B-cells to produce an anti-ganglioside response against human peripheral nerves. Humoral response frequently occurs as a result of the previous processes, resulting in blurred distinction between self and non-self cells.^{7,8}

The pathology of the disease process causes inflammation and destruction of the myelin (white matter) supporting the spinal cord, swelling and inflammation of the spinal cord tissue (grey matter), and results in scarring within and around the spinal cord itself. While the inflammatory process causes the acute symptoms that lead to

Table 1.

Criteria for the diagnosis of Idiopathic Transverse Myelitis (Adapted from: Transverse Myelitis Consortium Group. Barnes, G, et al. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology 59:499-505, 2002.)

Inclusion Criteria	Exclusion Criteria
Development of sensory, motor, or autonomic dysfunction attributable to the spinal cord	History of prior radiation to the spine within ten years
Bilateral signs and/or symptoms (not necessarily symmetric)	A clear distribution of clinical deficits consistent with anterior spinal artery thrombosis
Clearly defined sensory level	Abnormal flow voids on the surface of the spinal cord that could be consistent with arteriovenous malformations
Exclusion of extra-axial compressive aetiology by neuroimaging	Serologic or clinical evidence of a systemic autoimmune disease
Inflammation in the spinal cord demonstrated by CSF pleocytosis, elevated IgG or gadolinium enhancement on MRI within the first seven days	CNS manifestation of an infectious cause
Clinical progression to nadir between 4 days and 21 hours after onset	Brain lesions on MRI that are suggestive of multiple sclerosis
	Prior history of optic neuritis

Table 2.
Subclasses of Transverse Myelitis.

Subclass	Imaging Findings	Presentation
Acute Flaccid Myelitis	Bilateral, symmetric and widespread lesions in the grey matter at the affected level on MRI	Neurological disease that manifests with clinical syndromes similar to poliomyelitis
Acute partial transverse myelitis	Mildly or grossly asymmetric with an MRI lesion extending one or two vertebral segments	Spinal cord dysfunction causing symmetric, complete (or near complete) neurological deficits (paresis, sensory loss, and autonomic dysfunction) below the level of the lesion that onsets between 4 and 21 days. Signs and symptoms may include pain, weakness, uncoordinated movements, numbness, dysaesthesia, bowel and bladder dysfunction, sexual dysfunction depending on level affected
Acute complete transverse myelitis	Mildly or grossly asymmetric lesions extending more than one to two vertebral segments on MRI	
Longitudinally extensive transverse myelitis	Mildly or grossly asymmetric lesions that extend three or more vertebral segments on MRI.	

the diagnosis, the lasting pathology is thought to be as a result of the scarring within the spinal cord interfering with nerve signalling.

The classic symptoms of this disease exist on a continuum of myelopathy that present over the course of days to weeks. This most commonly includes back pain (30-50%), paraparesis (50%), lower limb paraesthesiae (80-95%), allodynia (80%), sensory level changes (80%), and bladder symptoms (nearly 100%).^{1,2,9} TM most commonly occurs in the thoracic spine, although there is no clear reason for this.

The current diagnostic criteria was completed in 2002 (see Table 1).⁹ The recommended approach for the diagnosis of TM should be based off the principle of exclusion including: the patient's clinical presentation, spinal magnetic resonance imaging (MRI), and serological evaluation, cerebrospinal fluid evaluation, neuroimaging, and possible others (i.e. positron emission tomography, biopsy) to exclude other differential diagnoses.^{3,6,9}

The presentation and prognosis of TM differs greatly, with some patients recovering with little to no problems, while others experience permanent impairments that have a large impact on their quality of life, and ability to perform their activities of daily living.^{1,10} There are three sub-classes of transverse myelitis that are delineated based on severity of symptoms and longitudinal extent of involvement in the spinal cord (see Table 2).^{2,3}

Given the array of symptoms that are possible with TM, it is crucial to get advanced imaging of the spine within three weeks to help rule out other possible differentials diagnoses and direct treatment accordingly. Differ-

ential diagnoses include vascular myelopathies, vitamin deficiencies (vitamin B12, vitamin E), and neoplasms.³ In those who are not aware of the possible implications, this array of symptoms can lead to misdiagnosis, delay in diagnosis, and in turn, delayed recovery.

According to sources,^{1,3,11-13} common medical management is to prescribe high dose steroids and immunosuppressants to mitigate the acute inflammation process from occurring if possible. High dose intravenous steroids and immunosuppressants should be started as soon as possible, as they are effective in acute inflammatory central nervous system diseases like TM, MS, and Guillain-Barre Syndrome.^{12,13} There is no standard approach or framework for conservative management and rehabilitation of transverse myelitis, and most practitioners rely on guidelines for other spinal cord pathologies.¹² Incorporating aggressive physical therapy and rehabilitative exercises appear to improve patient function and prognosis in the long-term.^{1,10}

The prognosis for TM is variable, though most with idiopathic TM can expect at least partial recovery.¹⁴ Symptoms that onset rapidly, and those who are younger at onset, tend to have a poorer prognosis.^{1,10} Recovery can be anticipated if significant progress is made in the first three months, however, 40% will have persistent morbidity.^{10,14} Common contributors to morbidity include motor weakness, paraesthesia, bowel and bladder dysfunction, and pain.

Chiropractors and other rehabilitation providers would be remiss if they do not perform and record through baseline neurological evaluations and compare regularly in

future visits to prevent missed recurrences.³ Most patients will have a monophasic disease process, while 20% may will have recurrent inflammatory episodes within the spinal cord.⁸ There is limited preliminary evidence that in approximately 5% of cases, patients with acute complete TM may progress to MS, further underlining the value of regularly tracked neurological evaluations.

Case Presentation

A 61-year-old cattle farmer presented to a rural chiropractic clinic in April 2017 for an initial evaluation. He did not smoke or use recreational drugs. He had no history of systemic illness or recent infection. There were no signs of neurological involvement or red flags, though there was a history of a C4-6 anterior fusion of the cervical vertebrae due to anterolisthesis performed in 1997. The physical exam included a neurological screen (deep tendon reflexes, motor testing of the upper and lower limbs, and crude touch sensory testing of the upper and lower limbs, and straight leg raise) which was normal, as well as appropriate orthopaedic testing of the involved joints and muscles. He was diagnosed with mechanical neck and low back pain. He was treated twice with spinal manipulation, soft tissue therapy and rehabilitative exercises. The managing chiropractor (JC) went on maternity leave in April 2017. In May 2017 she received an email from the patient's wife indicating for the past ten days he had experienced 'very bothersome' back pain and was having difficulty walking properly and coordinating his feet. In late April, he missed steps and fell down the stairs at home, which alarmed him greatly. He reported having interscapular pain and numbness bilaterally in the fingers. The patient reported the most aggravating behaviour was getting in and out of cars and chairs, and when he would relax in the evening, his left arm and leg would jerk involuntarily. If he were to get onto the ground to work with his calves while on the farm, he would be unable to stand again. Due to concern about the surgical plate, they went to their medical doctor (MD). After a brief examination that did not include a neurological screen, the doctor suggested it was likely viral and advised him to rest and wait at home for a week.

According to emails exchanged at this time and the patient's diary, the patient and his wife went to a new chiropractor in the interim in hope of relief from the interscapular and low back pain. The chiropractor also did not perform a neurological screen, yet treated him with

interferential current to the low back, traction to the low back, manual and drop piece adjustments. After the initial treatment, the patient reported feeling improvement for two to three hours before all of the symptoms returned, including feeling 'cold on the inside' of his now mostly flaccid right leg. The chiropractor saw the patient three more times that week, changing the diagnosis from mechanical back pain to 'a locked hip'. In the meantime, the initial chiropractor (JC) offered to contact the family doctor to arrange interprofessional collaboration, which was denied by the patient citing minor improvements in the leg pain. He would quickly deteriorate throughout that day and would be in severe pain throughout the shoulders, low back, and left leg. The numbness in the fingers had now spread proximally to include the hands. He also began to lose control of his bowels and experience overflow incontinence. By late May, after very regular care, the second chiropractor advised the patient to return to the MD as there was no improvement in his condition and new symptoms suggesting cauda equina syndrome. This was three weeks from the initial presentation at the chiropractor's office, and four weeks from the onset of symptoms. The timeline of the patient's presentation has been compiled in the table below from the patient's wife, his diary, and the medical reports from the hospital that treated him (Table 3).

The MD did a thorough neurological exam and wrote an urgent neurology referral allowing the patient to see a neurologist in late May. The subject had an MRI of the thoracic spine without contrast that found no bony anomalies, but found moderate degenerative changes (multilevel osteophytes causing foraminal stenosis) and an osteochondral bar mildly indenting the spinal cord assumed to be from the cervical fusion performed in 1997. Further, there was a high T2 signal in the thoracic spine at T5 and T6 suggestive of edema. From the neurologist notes, the patient had normal upper limb neurological testing. Lower limb testing revealed motor weakness (4/5 bilaterally), brisk hyperreflexia (3+ bilaterally), and an upgoing plantar response. The neurologist sent him for plain film imaging of the cervical and thoracic spine and a full spine MRI on the same day. Based on the subject's imaging and presentation, the neurologist notified the family to prepare for emergency surgery the next morning to remove the osteochondral bar and sent him to the emergency department for an MRI with gadolinium contrast and ce-

Table 3.
Timeline of the patient's presentation.

Month	Notable event	Outcome
April 2017	Original chiropractor (JC) initial assessment	Mechanical back pain.
May 2017	Email from spouse re: new onset symptoms	Advised to go to MD, who suggested it was viral. Went to a second chiropractor instead.
May 2017	Original chiropractor offered to notify MD of severity of symptoms	Patient denied, citing it was feeling better. Continued being seen by second chiropractor.
May 2017	Second chiropractor referred back to family MD	Full neurological assessment, urgent neurological referral.
May 2017	MRI without contrast	Degenerative changes, osteochondral bar, notable cord edema at T5-T6.
May 2017	Neurology consult	Plantar response upgoing bilaterally No clonus, no ataxia No bulbar or cranial deficits
May 2017	MRI with contrast Emergency neurology assessment	Two tiny enhancing foci in the T2 area on the anterior right lateral surface with decreased edema than May 29th, ruling out primary malignancy Upper extremity neurological exam normal Lower extremity: bilateral spasticity, no clonus, 3+ reflexes in lower limb (more prominent distally), 2- 3/5 strength in lower limb (left weaker than right) Plantar response upgoing bilaterally Good rectal tone and sensation Admitted for intravenous steroids (Solu-Medrol) while additional panels were being run (autoimmune, infectious, metabolic)
June 2017	Discharged	Notable improvement in sensation and motor function, not advised to continue medication at home and follow up with community neurologist.
June 2017		Began to notice symptoms develop over 2-3 hours while driving his tractor. Progressive weakness in leg over the next three days.
June 2017	Emergency neurology	Unable to ambulate Cranial nerves normal, cerebellar testing normal Unable to lift left leg off stretcher more than 10°, right leg could elevate to 45° Serological testing returned normal (CBC, electrolytes, BUN, creatinine, CRP, ESR, thyroid) Negative for lupus, ANA, ANCA, RF, infectious agent antibodies Cerebrospinal fluid was grossly normal Indication of progression of transverse myelitis Sent for contrast MRI of brain, cervical and thoracic spine
June 2017	Brain MRI without contrast Neurology	Non-specific white matter abnormalities in brain Considerable progression of the cervical cord lesion extending C2-C7 with significant cord edema throughout. No abscess or epidural collection. Thoracic spine remained stable with two high signal foci noted at T5 and T6. Left leg is plegic (1/5 motor), 3+ reflexes bilaterally, plantar response upgoing Upper limb strength is 3-4/5 bilaterally Bladder retention visualised, therefore a catheter was inserted Diagnosis of longitudinally extensive transverse myelitis rendered Patient re-started on steroids and intravenous immunoglobulin therapy.

rebrospinal fluid testing. The contrast MRI identified two tiny enhanced foci on the right anterior spinal cord, with reduced edema compared to the MRI performed the day prior. The differentials at this point were broad, including metabolic, infectious, and autoimmune-related TM, MS, sarcoidosis, and paraneoplastic disease. The imaging identified it the patient did not have a compressive lesion in the spine, therefore he did not receive surgery. Instead, he was put on high-dose intravenous corticosteroids to try to reduce the inflammation seen in his spinal cord. He responded well to the steroids and was discharged in early June to his community neurologist. Within three days his symptoms began to onset again after driving his tractor. He reported sensory disturbances, but denied incontinence or vision problems. He returned to the emergency department four days later, unable to walk. On evaluation, his left leg was plegic and both legs were spastic. He had begun to develop symptoms of upper motor weakness. Serological testing, cerebrospinal fluid testing, cerebellar testing and cranial nerve testing were all within normal limits. He was sent for a follow-up MRI and by the middle of June was diagnosed with longitudinally extensive transverse myelitis of idiopathic origin having ruled out neoplastic and auto-immune causes, and with no evidence of infectious agents. He was re-started on intravenous steroid therapy and started intravenous immunoglobulin (IVIG) therapy. Approximately one week after receiving his diagnosis, the clinical note indicates that despite the IVIG and steroid therapy, the patient's presentation was progressively declining. At this point, he was plegic bilaterally and developing bilateral arm weakness and sensory loss. He was reliant on a catheter. Due to his lack of response to high-dose steroids, a referral was made to begin a new treatment of plasmapheresis at St Michael's Hospital in Toronto. Three days later the patient was transferred. Another contrast-enhanced MRI study was performed as well as a lumbar puncture, of which the results are not available to us. He was started on an immunosuppressant, Imuran, while continuing with steroid injections through the intravenous route, discontinuing IVIG therapy. Shortly after the middle of July, the patient was transferred to a spinal cord rehabilitation hospital. His diagnosis changed to acute complete transverse myelitis.

The patient was permanently wheelchair bound. He was reliant on a catheter, but did not need respiratory assistance. Over the next two years, the patient experienced

a number of complications including autonomic dysreflexia, pneumonia, osteomyelitis, bed sores, hypotension, urinary tract infections, dysphagia, and chronic pain. The family relied heavily on community support as he was no longer able to work as a farmer. He lost nearly 100lbs and developed contractures in his shoulders, hips, and knees. He was able to move his left arm minimally, and lost use of the rest of his body below the site of the lesion in the cervical spine. He developed disuse atrophy in his left arm. Due to the frequency and duration of hospital stays from the complications, he was unable to receive regular physical rehabilitative therapy, nor care of his chronic pain and musculoskeletal complaints. The subject passed away from a host of complications in October 2019.

Discussion

According to the National Multiple Sclerosis Society, in >50% of cases the cause of TM is unknown.¹ It is thought to be caused by either an autoimmune response, bacterial or viral infection, or an active demyelinating disease process.¹⁻³ The signs and symptoms can present variably, within hours or over three weeks, which can significantly vary the prognosis. The presentation is bimodal (10-20 years, 30-39 years), which does not include our 61-year-old patient. As highlighted by Wei *et al.*¹⁵ in 2019, collaboration and communication among healthcare providers is essential in creating a synergy to provide efficient, safe, and high-quality patient care. While the patient's MD in this case did not appear to do an appropriate neurological exam initially, it is possible that the signs and symptoms were not developed enough to give the MD an indication of neurological involvement. The authors feel a neurological exam was pertinent for the chiropractor to perform given the new patient status, as well as the symptoms described. Ideally, if the chiropractor had been regularly monitoring this patient's neurological signs and symptoms, the concerning progression would have warranted prompt and urgent contact with his MD within the appropriate time frame (within three weeks) for aggressive steroid intervention. Further, if the chiropractor had notified the MD immediately, the MD may have identified that the symptoms were not resolving within the week as he had anticipated and called the patient in to his office to commence further evaluation. The five-week delay in referral to a neurologist prevented early assessment and diagnosis for this patient, likely worsening his prognosis.

sis. Having a prompt and complete evaluation of cases presenting as myelopathy ensures that idiopathic TM is differentiated from other possible diagnoses in a timely manner to allow appropriate treatment of spinal cord edema, possibly reducing symptom severity and disease progression.^{9,11} It is unclear in this case how much of an impact the delayed diagnosis had on the patient's disease severity and outcome.

There is no cure for TM¹, and while there are some proposed approaches for the treatment of this condition, there are no clinical guidelines for its acute or long term management^{1,11}. The current standard of treatment includes initially treating with corticosteroids and immunotherapy in the acute phase of TM, with the goal to stop the progression and help decrease the inflammation at the spinal cord lesions.^{1,3,11,12} Intravenous corticosteroids are the first-line of treatment, despite no randomized controlled study to support its effectiveness, this approach is backed by clinical experience, and evidence from related disorders.^{11,12} If there is minimal improvement with corticosteroid use after five to seven days, or the patient has moderate to severe TM, plasma exchange is often used.¹³

Many patients require long term interdisciplinary management to address the spectrum of associated concerns of living with TM including physical rehabilitation, mental health, support for activities of daily living, accessible work and living spaces, and more.^{1,10} In this case, it appears that there was minimal collaboration between any of the health care professionals involved prior to hospitalization. In cases such as these when non-specific clinical findings indicate a CNS pathology, chiropractors have a specific and important role: to identify it and refer immediately. The authors believe the patient's outcome would have been improved if the chiropractor opened lines of communication early in this patient's management. All patients affected with TM have notable physical and psychological challenges that require a full team of allied healthcare providers to address. Evidence suggests that early and aggressive rehabilitation therapy improves patient outcomes, though 40% will have persistent morbidity.¹⁰ Manual therapists, such as chiropractors, can help in the rehabilitation process by improving joint range of motion, and through the prescription of stretching and strengthening exercises to allow for independent completion of activities of daily living.¹⁶

If the diagnosis of TM is rendered early in onset, it is

conceivable that early intervention would decrease the severity and extent of spinal cord inflammation. Chiropractors are portal-of-entry practitioners and are well-suited to screen for and appropriately refer patients who appear to have a neuropathological presentation.

Summary

This study aims to provide an update in the current state of evidence of TM, as well as the role of interprofessional collaboration in patient care. The presentation and prognosis of TM differs greatly, with some patients recovering with little to no problems, while others experience permanent impairments that have a large impact on their quality of life, and ability to perform their activities of daily living. Practitioners require increased awareness of the presentation of spinal cord myelopathies to decrease misdiagnosis and/or diagnostic delay. Health care providers are remiss if they do not perform a neurological examination with each new patient. An accurate and time efficient diagnosis is the key to providing appropriate treatment methods to help minimize long term deficits. While current therapies are largely non-specific, it is important to have an established circle of care for health professionals to communicate openly about patient evaluation and management.

References

1. National Institute for Neurological Disorders and Stroke. Transverse Myelitis Fact Sheet NIH Publication 17-4841[Internet]. 2019 Aug[Cited 2019 Oct 30]. Available from:www.ninds.nih.gov/disorders/patient-caregiver-education/fact-sheets/transverse-myelitisfact-sheet/
2. Beh SC, Greenberg BM, Frohman T, Frohman EM. Transverse myelitis. *Neurol Clin*. 2013; 31:79.
3. Krishnan C, Kaplin AI, Deshpande DM, Pardo CA, Kerr DA. Transverse myelitis: pathogenesis, diagnosis and treatment. *Front Biosci*. 2005;9(1483): 99.
4. Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, De Seze J, Fujihara K, Greenberg B, Jacob A, Jarius S. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurol*. 2015; 85(2): 177-189.
5. Jacob A, Weinshenker BG. An approach to the diagnosis of acute transverse myelitis. *Semin Neurol*. 2008; 28(1): 105-120.
6. Zalewski NL, Flanagan EP, Keegan BM. Evaluation of idiopathic transverse myelitis revealing specific myelopathy diagnoses. *Neurol*. 2018. 90(2): e96-102

7. Rodríguez Y, Rojas M, Pacheco Y, Acosta-Ampudia Y, Ramírez-Santana C, Monsalve DM, Gershwin ME, Anaya JM. Guillain–Barré syndrome, transverse myelitis and infectious diseases. *Cell Molec Immunol*. 2018;15(6): 547-562.
8. Kaplin AI, Krishnan C, Deshpande DM, Pardo CA, Kerr DA. Diagnosis and management of acute myelopathies. *Neurologist*. 2005; 11: 2–18.
9. Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurol*. 2002; 59(4): 499-505.
10. Defresne P, Hollenberg H, Husson B, et al. Acute transverse myelitis in children: clinical course and prognostic factors. *J Child Neurol*. 2003; 18: 401.
11. Frohman EM, Wingerchuk DM. Transverse myelitis. *N Engl J Med*. 2010; 363(6): 564-572.
12. Scott TF, Frohman EM, De Seze J, Gronseth GS, Weinshenker BG. Evidence-based guideline: clinical evaluation and treatment of transverse myelitis: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurol*. 2011; 77(24): 2128-2134.
13. Awad A, Stuve O. Idiopathic transverse myelitis and neuromyelitis optica: clinical profiles, pathophysiology and therapeutic choices. *Curr Neuropharmacol*. 2011; 9(3): 417-428.
14. Young J, Quinn S, Hurrell M, Taylor B. Clinically isolated acute transverse myelitis: prognostic features and incidence. *Mult Scler*. 2009; 15(11): 1295-1302.
15. Wei H, Corbett RW, Ray J, Wei TL. A culture of caring: the essence of healthcare interprofessional collaboration. *J Interprof Care*. 2019; 34(3): 324-331.
16. Sadowsky CL, Becker D, Bosques G, Dean JM, McDonald III JW, Recio A, Frohman EM. Rehabilitation in transverse myelitis. *CONTINUUM: Lifelong Learning in Neurology*. 2011; 17(4): 816-830.